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GEOPHYSICAL VARIABLES AND BEHAVIOR: LXXXV. SUDDEN INFANT DEATH, BANDS OF GEOMAGNETIC ACTIVITY, AND PC1 (0.2 TO 5 HZ) GEOMAGNETIC MICROPULSATIONS[1]

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Summary.—Pc1s (continuous pulsations) within the geomagnetic field, whose durations are about 30 minutes but which can reoccur several times nightly, are observed during periods when global geomagnetic activity is very low (less than 10 nT). The hypothesis that these 0.2 to 5 Hz synchronized micropulsations or hydromagnetic emissions might stimulate physical chemical cascades within the brain that precipitate the sudden death in infants was tested by correlational analysis for a two-year period (1960–1961) for Ontario. Results were consistent with the hypothesis that the monthly incidences of these unexpected deaths, pc1 micropulsations and geomagnetic activity less than 10 nT displayed a shared source of variance. Implications are discussed.

The etiology for the sudden and unexpected death of the majority of infants who die between one month and one year of age has been attributed to Sudden Infant Death (Kinney, Filiano, & Harper, 1992). These deaths occur primarily around three months of age, during the early morning hours and on autumn-winter days. On the basis of the temporal clustering of these unexpected deaths over large geographical areas, the conspicuous diurnal and seasonal patterns, and the postmortem histomorphological measurements, O'Connor and Persinger (1997) hypothesized that the primary spatially pervasive variable responsible for sudden deaths of infants could be some specific feature of geomagnetic activity. A similar hypothesis was first developed by Eckert (1992).

The earth's magnetic field contains a steady-state component, which is within the range of 50,000 nT and time-varying components (periods >1 sec.) which are in the range of 1 to 500 nT. The typical measures of the time-varying component, or geomagnetic activity, are 3-hr. or 24-hr. averages of the amplitudes of the most disturbed component of one of three spatial inferences of the geomagnetic field from several stations that represent the earth's surface; however, these measures do not reflect the frequency bands of the variations in intensity. The situation is analogous to measuring only the microvoltage change on a meter during an electroencephalographic recording while ignoring the frequency. In this instance the slow increase and then decrease in potential differences as the brain states shift to light sleep

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would not show the shifts from beta frequency to alpha rhythms and then to low amplitude slower mixed frequencies containing sleep spindles.

Traditional correlational analyses such as those between the intensity of the geomagnetic variation (the aa or antipodal index) and the magnitude or numbers of many biobehavioral phenomena (Michon & Persinger, 1997) would not be significant because of the intrinsic nonlinearity between intensity and the shifts in qualitative features (the time varying morphology) over the intensity range. O'Connor and Persinger's (1997) results confirmed that partialling months into the numbers of days that contained days whose average geomagnetic activity occupied successive aa increments of 5 nT could explain most of the variance in the monthly occurrence of numbers of sudden infant deaths for a 2-yr. period.

We have considered the possibility that increments of geomagnetic activity could be associated with different primary frequencies or wave shapes whose biological effectiveness would be distinct. If the brain is an electromagnetic matrix that generates aggregates of responses, then there may be a large domain of evolution-selected, specific geomagnetic stimuli that might evoke basic intrinsic neuroelectromagnetic patterns that would produce these responses. These geomagnetic stimuli presented over time might be considered the electromagnetic equivalents to the specific DNA sequences within space that produce microstructural changes within the cell.

In the present study we tested the hypothesis that pc1 geomagnetic pulsations (continuous pulsations around 1 sec.), also described as hydromagnetic emissions or pearls, may be a candidate environmental stimulus which could contribute to the physiochemical mechanisms hypothesized to result in sudden deaths of infants. These pulsations are regular sinusoidal forms with a beating character that can be described as a "string of pearls" when measured by strip-chart recorders (Campbell, 1967). The midfrequency of pc1 pulsations or pearls range between 0.2 to 5 Hz whose amplitudes are usually less than 0.1 nT. They are generated as hydromagnetic emissions from the upper magnetosphere and move to the surface by conductive ducts (Antonova, Fligel, & Dovbnja, 1981).

Although the duration of an episode is about 0.5 hr. (on rare occasions up to 12 hr.), there can be regularly recurring groups for several hours during very active days. High activity of pc1s usually appear in groups or clusters of two to five successive days. Pc1s occur primarily between 01 hr. to 04 hr. local time and are more prominent during solar quiet periods within 5 to 7 days after an isolated geomagnetic storm (Heacock & Akasofu, 1973). The midfrequency is highest during postmidnight hours and lowest during local afternoon. Their intensity can be enhanced by spread-F, which has been attributed to irregularities in geomagnetic-field-aligned electron densities within the F-region of the ionosphere (Fraser-Smith, 1972). These irreg-

ularities may extend for hundreds of kilometres over the earth's surface. The occurrence of pc1s has been shown to have an enhancement on weekends (Fraser-Smith, 1979) which has been hypothesized to reflect an interaction with the industrial usage of power frequencies.

Method

We obtained the monthly occurrences of days of hydromagnetic emissions (pc1 pulsations) in California, the closest geographical measurements, from Fraser-Smith (1970). The definition of a pc1 day was a 24-hr. U.T. day containing one or more pc1 pearl pulsation events (Fraser-Smith, 1981). These values were added to our previous database that contained the numbers of days per month that contained successive increments of daily, global geomagnetic activity, as defined by the aa index, between 0 and 5 nT, 6 and 10 nT, 10 and 15 nT, . . . (O'Connor & Persinger, 1997). Correlational, factor and partial correlation analyses were completed with SPSS software.

Results

Zero-order correlations between the monthly incidence of pc1s and the numbers of days whose average values were within the various 11 5-nT increments showed a significant ($p < .05$) positive correlation ($rho = .63$) for the numbers of days with average aa values between 1 and 5 nT but significant negative correlations between the monthly incidence of pc1s and days with values between 6 and 10 nT (–0.50) and 15 and 20 nT (–.67). However, the correlation between the monthly incidence of sudden deaths of infants and pc1s was not significant ($rho = -.24$).

The zero-order parametric correlations between monthly incidences of sudden infant deaths, days with values between 1 and 5 nT and pc1s were .39 for the first two, .42 for the latter two, and –.14 for the first and last variables. The partial correlation between the monthly incidence of pc1s and numbers of infant deaths was –.37 when the shared variance with the numbers of days whose average aa values ranged between 1 and 5 nT was removed. When the shared variance between the monthly incidence of pc1s was removed from both the values for the numbers of sudden infant deaths and days with values between 1 and 5 nT, the partial correlation between the two was .50 ($df = 9$, $p < .05$). These results were consistent with the hypothesis that numbers of sudden deaths of infants increased during those months that contained more very quiet days with values between 1 and 5 nT.

An exploratory factor analysis (because of the small sample size) involving values for the numbers of infant deaths, pc1s and days with values from 1 through 5 nT, 5 through 10 nT, 11 through 15 nT, and 16 through 20 nT, indicated significant loadings on the first factor (eigenvalue = 2.75; explained variance 39%) for pc1s (0.44), days with values between 1 and 5 nT (.91), 6

through 10 nT (.81), and 15 through 20 nT (–.79). The numbers of infant deaths was not significantly loaded ($r = .31$) on this factor.

Given the strongly seasonal component (monthly values ranging from 4 to 18) in the numbers of sudden infant deaths, which was evident even in this database, the effects of season were first removed. When this was completed, the partial correlation between the monthly incidence of infant deaths and pc1s was .58 ($df = 9$, $p < .05$). Visual inspection of the scattergram of the correlation between the residuals (the predicted minus the observed values with the linear order of month as the independent variable) verified that the relationship was not due to outliers. The range of the residuals for the numbers of infant deaths was six and would suggest that approximately one-third of these cases was associated with pc1s after the effects of season had been removed. Factor analysis of the residual for pc1s, the residual for numbers of infant deaths and number of days with geomagnetic aa values for the four intervals showed that, after varimax rotation, the incidence of pc1s (.84), number of deaths (.70), days with increments between 1 and 5 nT (.53), and between 15 through 20 nT (–.80) were loaded on the same factor (eigenvalue = 3.25, 46% of variance).

Discussion

These results would be consistent with the hypothesis that pc1 pulsations or hydromagnetic emissions could be a significant environmental stimulus that triggers the physiochemical mechanisms within the brain that result in clusters of the sudden and unexpected deaths of infants. The loading of the monthly numbers of these deaths on the same factor as that associated with the monthly incidence of pc1s and low bands of geomagnetic activity would be expected if they shared the same source of variance. Because the data involve monthly incidences, we do not know the actual temporal contiguity in days between the occurrence of the geophysical events and the sudden deaths of infants.

The role of pc1s in the precipitation of sudden deaths of infants would be consistent with the tendency for both to occur in temporal clusters and to occur over large areas. The relationship between spread-F and pc1s indicates that large areas under these large ionospheric "lens" would be more susceptible to the occurrence and density of pc1s. There are several predictions about the temporal and spatial clusters of sudden deaths of infants that would support this argument. For example, if micropulsations increase on weekends due to decrease in the cultural use of power frequencies (Fraser-Smith, 1979), then there should be a significant increase in the incidence of sudden deaths of infants during Saturday and Sunday compared to midweek. Considering the full-moon enhancement of hydromagnetic emissions other minor hypotheses could be tested to discern the specificity of the rela-

tionship between the daily incidence of pc1s and infant deaths as a function of lunar phase and distance.

Whether only specific infants are vulnerable to these stimuli or *any* infant would be vulnerable (and this propensity would be determined by the probabilistic congruence between the occurrence of pc1s, their focus into an area, and the resonance with the infant's brain activity), must still be discerned. Experimental data collected 25 years ago (Persinger, 1973) for adult rats during near-cardiac failure from successive dosages of pentobarbital indicated that "cardiac driving" occurred around 0.5 Hz to 1 Hz rather than normal cardiofrequency of 3 to 6 Hz. Recent data indicate that baroreceptors, which are the major input to the central cardiovascular pathways, are tuned to inputs of about 1 Hz through 10 Hz (Fan, Reynolds, & Andresen, 1996). These receptors modulate the neuroelectrical activity that ascends from the parasolitary nucleus (brain stem) into the central amygdala and insula and then descends from these two "oscillating pathways" through the hypothalamus into brain-stem centers. These same areas have been implicated in sudden cardiac death (Persinger, 1995).

The weak intensity of the band of pc1s, whose amplitudes are in the order of 0.1 nT to 1 nT, is often the primary argument employed by classical biophysicists to dismiss the role of these natural phenomena in biological processes. If pc1s are effective stimuli, then the brain of the infant must be able to amplify the amplitude of these frequencies in a manner similar to the progressive recruitment of unstable neurons by a single neuron into a massive proxysmal discharge. The young infant would be vulnerable because the paucity of inhibitory neurons and the decreasing taurine levels (an intrinsic electrically dampening amino acid that displays the greatest concentrations in very young infants) do not afford the protection of the ontogenetically older brain.

Consequently, large populations of neurons could be synchronized to exhibit calcium oscillations. The synchronization by the specific *temporal pattern* of magnetic field oscillations (the electromagnetic analogue of a ligand or its antagonist) of calcium waves within large aggregates of cells would effectively paralyze the nitric oxide system and result in death. Brief application of elementary-shaped (sine-wave) magnetic fields perturb nitric oxide-dependent neuronal activity. These magnetic fields appear to perturb nitric oxide production by affecting the calcium resonance and hence the frequency modulation of the calcium waves. We suspect that even lower intensity magnetic fields with more complex temporal morphologies which imitate natural phenomena may perturb the production of nitric oxide to the state of inhibition or suppression.

Bawin, Satmary, Jones, Adey, and Zimmerman (1996) have supported this assumption. They found that continuous application of a weak, 1-Hz

magnetic field resulted in an irreversible destabilization of rhythmic slow potentials (theta) in rat hippocampal slices mediated by a nitric oxide-dependent process. The role of the hippocampus in compensatory respiratory control, as demonstrated by the appearance of enhanced theta activity in apnea before the recovery of breathing (Harper, Poe, Rector, & Kristensen, 1998), suggests that destabilization of rhythmic potentials would compromise recovery from hypoxic episodes. If a similar phenomenon works naturally, then clusters of infant deaths should occur during or shortly after successive days of geomagnetic pulsations.

This model would also be consistent with our previous hypothesis that the sudden suppression of circulating levels of melatonin from the pineal organ by specific bands of geomagnetic activity increase the risk of sudden death. The suppression would transiently increase the electrical lability within brain regions in general and in those that control the cardiovascular system in particular. Reduced production of melatonin, an inhibitor of nitric oxide synthase (Pozo, Reiter, Calvo, & Guerrero, 1997), could amplify the effective coupling between the ambient geomagnetic field and neuronal activity. The desynchronization of the two descending pathways to the solitary nucleus of the brain stem would precipitate a sudden cessation of cardiac activity. Any condition modifying nitric oxide synthase levels, from maternal smoking (Ulm, Plockinger, Pirich, Gryglewski, & Sinzinger, 1995) to "developmental windows," e.g., 2 mo. to 6 mo. during ontogeny with its unique but transient brain microstructure, would increase the risk.

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